PB85-243137

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(U.S.) Environmental Research Lab.
Gulf Breeze, FL

Jul 85

U.S. Department of Commerce
National Technical Information Service
NTIS

PB85-243137

EPA/600/D-85/167
July 1985

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by

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1. REPORT NO. EPA/600/D-85/167	2.
3. RECIPIENT'S ACCESSION NO. PB85 243137/AS	
4. TITLE AND SUBTITLE ASSOCIATIONS BETWEEN PHYSIOLOGICAL ALTERATIONS & POPULATION CHANGES IN AN ESTUARINE MYSID DURING CHRONIC EXPOSURE TO A PESTICIDE	5. REPORT DATE July 1985
	6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S) Charles L. McKenney, Jr.	8. PERFORMING ORGANIZATION REPORT NO.
9. PERFORMING ORGANIZATION NAME AND ADDRESS	10. PROGRAM ELEMENT NO.
	11. CONTRACT/GRANT NO.
12. SPONSORING AGENCY NAME AND ADDRESS U.S. Environmental Protection Agency Environmental Research Laboratory Office of Research & Development Gulf Breeze, FL 32561	13. TYPE OF REPORT AND PERIOD COVERED
	14. SPONSORING AGENCY CODE EPA/600/04
15. SUPPLEMENTARY NOTES	

16. ABSTRACT

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17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group

18. DISTRIBUTION STATEMENT Release to public	19. SECURITY CLASS (This Report) Unclassified	21. NO. OF PAGES 25
	20. SECURITY CLASS (This page) Unclassified	22. PRICE

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE

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ASSOCIATIONS BETWEEN PHYSIOLOGICAL ALTERATIONS AND POPULATION CHANGES IN AN ESTUARINE MYSID DURING CHRONIC EXPOSURE TO A PESTICIDE

Charles L. McKenney, Jr.

SUMMARY

A number of vital life processes of an estuarine mysid (Mysidopsis bahia) were examined throughout its life cycle during exposure to the thiocarbamate herbicide, thiobencarb. Initial exposure of juvenile mysids to thiobencarb resulted in elevated respiration rates. Concentrations of thiobencarb, that produced significant reductions in population survival through a complete life cycle (approximately 24 d), significantly stimulated respiration rates of juveniles after only 4-d exposure. Increased metabolic demands with sublethal thiobencarb exposure reduced the amount of assimilated energy available for production of new tissue by juvenile mysids, resulting in retarded juvenile growth rates. Modifications in the energy metabolism of individual mysids exposed to thiobencarb were related to decreases in total young production of discrete populations. Greater usage of proteinaceous substrates for energy metabolism during the maturation of M. bahia was altered by exposure to high sublethal concentrations of thiobencarb. Higher O:N ratios during the maturation of these thiobencarb-exposed mysids suggest a greater reliance on the more energy-rich lipid substrates to support the elevated rates of oxidative metabolism, resulting in less lipid material being available for gamete production.

INTRODUCTION

To adequately assess the biotic hazard of contaminants entering the marine environment, suitable criteria must be determined for detecting the ecological damage resulting from long-term exposure to low concentrations of contaminants. While it is generally recognized that lower levels of biological organization (cellular and organismal) respond to environmental stress long before higher levels (community and ecosystem), the ecological significance of biotic responses at lower levels may be difficult to interpret (Waldichuk, 1979; Bayne et al., 1980; Kinne, 1980; Sastry & Miller, 1981). However, for a number of years ecologists have examined the productive processes of communities and ecosystems by studying the underlying physiological patterns of response of a species throughout its life cycle to environmental variables (Mann, 1969; Winberg, 1971; Steele, 1973, 1974; Grodzinski et al., 1975). Application of these concepts of ecological energetics to the field of aquatic toxicology may provide valuable "tools" for use both in the laboratory as short-term predictors of chronic effects and in the field for monitoring long-term ecological effects of reduced water quality.

In the last decade, a mysid shrimp (Mysidopsis bahia) has been shown to be very sensitive to a variety of toxic substances when compared with other marine species (for a review see Nimmo & Hamaker, 1982). The responses of this zooplankter to chronic exposure to microcontaminants (including increased mortality in the population, retarded growth, and diminished reproductive success) have direct effects on the population production of the species, which serves as an important link in the marine food chain between primary producers and fish. Furthermore, a recent study (McKenney, 1982) demonstrated the interrelationships between energy metabolism, growth dynamics, and reproduction of M. bahia exposed through an entire life cycle to the chlorinated organic pesticide, endrin. Increased metabolic demands for maintenance during endrin exposure reduced the growth efficiency of juveniles, resulting in retarded somatic production rates. At the same sublethal concentrations of endrin, increased lipid catabolism of maturing juveniles led to reduced reproductive capacity.

The objective of this study was to gain further understanding of the ecological consequences of physiological effects of toxic organics on a sensitive estuarine zooplankton species. Dose-response relationships of individual

physiological functions were examined for various life stages of M. bahia exposed throughout its life cycle to the organic pesticide, thiobencarb, and these physiological responses were compared with production processes of the population. Individual physiological responses were combined into indices of bioenergetic and metabolic significance for the various life stages and their dose-response relationships evaluated on the basis of their ecological significance.

MATERIALS AND METHODS

Mysidopsis bahia was exposed to thiobencarb (S-[4-chlorophenyl] methyl diethylcarbamothionate) through an entire life cycle using a proportional diluter as described by Schoor and McKenney (1983). Every 3 min the diluter produced one liter of filtered sea water for each of six thiobencarb exposure concentrations and a carrier control, each containing an equal portion of triethylene glycol. Each liter was delivered to a glass aquarium divided into three replicate chambers. Sea water in the exposure system was maintained at 20 ± 2 $^{o}/_{oo}$ salinity and 25 ± 1°C.

Throughout the course of the study, thiobencarb concentrations for each exposure condition were determined weekly by gas chromatography in accordance with procedures described by Schimmel et al. (1983). The lower limit of detection was 0.50 µg/L, and recovery from fortified samples was 96%. Mean measured concentrations of thiobencarb and their standard deviations for all exposure conditions were <0.50 (carrier control), 22 ± 6, 35 ± 10, 76 ± 35, 181 ± 56, 345 ± 140, and 734 ± 48 µg/L.

Newly released juvenile M. bahia (<24 h old) were obtained from ovigerous females maintained in static, recirculating cultures at the same salinity/ temperature conditions as the exposure system. For the survival and reproductive portions of the study, 15 juveniles were pipetted into Nitex® baskets in each of the triplicate chambers. Additional groups of mysids were placed in separate baskets at each exposure concentration and subsampled for metabolic and growth measurements. Throughout the study, all groups of mysids were fed an abundance of freshly hatched Artemia nauplii daily.

Survival observations were made at each exposure concentration on four life stages: early juveniles (Day 1), juveniles (Day 4), advanced juveniles (Day 10), and young adults (Day 16). In addition, ovigerous females were

isolated in pairs with mature males within Nitex® brood cups. Daily records were maintained for the day of release of the first brood and number of young released in the first brood by isolated females.

On days representative of the various mysid life stages, respiration rates and ammonia excretion rates were determined for seven individuals of each life stage per exposure condition in accordance with methods described by McKenney (1982). To reduce variability in these physiological responses due to diurnal rhythms, respiration and excretion measurements were taken for the various life stages at the same time of the day. Even though mysids were last fed Artemia approximately 18 h prior to these metabolic measurements, food remained available to them until their isolation period in the respirometers. O:N ratios were calculated from the atoms of oxygen consumed to atoms of nitrogen excreted. In addition, dry weights of individual mysids from each life stage and thiobencarb concentration were converted into values of daily weight gain (μg dry weight/day), and the caloric equivalents of these growth rates and the respiration rates were used to produce bioenergetic terms of production (P) and daily maintenance costs (R) as described by McKenney (1982). These values were used to derive net growth efficiency values (K_2) (Winberg, 1971) for each mysid stage under each thiobencarb exposure condition by the formula:

$$K_2 = \frac{P}{(P+R)} \qquad (1)$$

Differences in biological responses between thiobencarb concentrations were analyzed by analysis of variance, using Dunnett's procedure for multiple comparisons of treatment means to the control mean (Zar, 1974). Relationships between biological responses and thiobencarb exposure concentrations were examined by linear regression analysis, while more complex relationships between biological responses and thiobencarb exposure concentration for the various mysid life stages were examined by multiple regression analysis (McNeil et al., 1975; Box et al., 1978).

The most complex model was a two-factor, second-order polynomial equation, in which the entire data set for a

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particular biological response was regressed on the quadratic function of day in life cycle and thiobencarb concentration:

$$\hat{Y} = b^0x^0 + b^1CONC + b^{11}CONC^2 + b^2DAY + b^{22}DAY^2 + b_{12}(CONC \times DAY) \quad (2)$$

where $\hat{Y}$ = the estimated biological response; x^0 = the intercept; CONC and DAY = the linear effect of thiobencarb concentration and day in life cycle, respectively; $CONC^2$ and DAY^2 = the quadratic effect of thiobencarb concentration and day in life cycle, respectively; CONC x DAY = the linear by linear interaction between thiobencarb concentration and day in life cycle; and b_0, b_1, etc. = the regression coefficients (Meyers, 1971, Alderdice, 1972). If this model best explained the data, response surface curves were generated at a range of levels of these two factors by the General Linear Model (GLM) and PLOT procedures available in the SAS statistical package (Barr et al., 1976).

RESULTS

Exposure to a range of thiobencarb concentrations from 35 to 735 µg/L produced differential survival rates in populations of Mysidopsis bahia through an entire life cycle. Four days of exposure to concentrations of 345 and 735 µg thiobencarb/L resulted in significant ($p<0.05$) mortality (23 and 100%, respectively) in the mysid populations. No other concentrations produced significant mortality with continued exposure through the juvenile phase of the life cycle (approximately 2 wk). Survival of M. bahia exposed through an entire life cycle (24 d) was linearly related to exposure concentration of thiobencarb (Fig. 1A) and measured thiobencarb concentrations above 76 µg/L resulted in significant reduction in survival.

Production of young in the first brood by isolated females was linearly reduced by thiobencarb in such a manner that no young were produced in 345 µg thiobencarb/L (Fig. 1B). Total young production in the first brood of the population, however, was related to thiobencarb concentration in a curvilinear fashion (Fig. 1C). Significantly fewer young were produced by populations exposed to thiobencarb concentrations of 35 µg/L and higher. This inhibition in reproductive success of the population was maximum at concentrations higher than 181 µg thiobencarb/L.

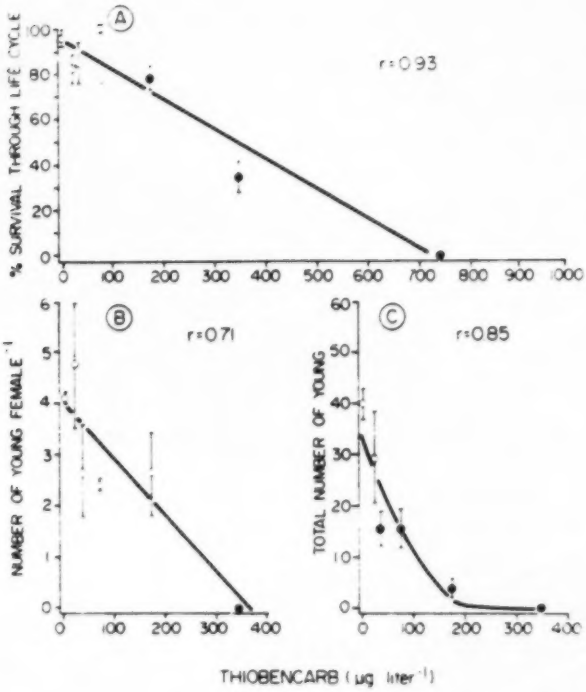

Fig. 1. (A) Survival percentage, (B) young production per female, and (C) total production of young by a Mysidopsis bahia population as a function of thiobencarb exposure through a complete life cycle. Values represent mean ± SE. Darkened circles are significantly different from control at 0.05 probability level.

Since a two-way analysis of variance demonstrated that mysid respiration was significantly ($p = 0.0001$) influenced by an interaction between life stage and thiobencarb concentration, respiration rates for individual life stages were regressed onto thiobencarb concentration (Fig. 2). As early as 24 h after exposure, mysid respiration was significantly ($p<0.05$) elevated by progressively higher concentrations of thiobencarb. As shown by the changing slopes and correlation coefficients (r-values) of the regressions, the linearity of this response increased with continued exposure through the juvenile phase of the life cycle. As juveniles matured and weight-specific respiration rate decreased, a

quadratic regression curving downward at the lower concentrations best explained the respiration response of adult mysids to thiobencarb exposure.

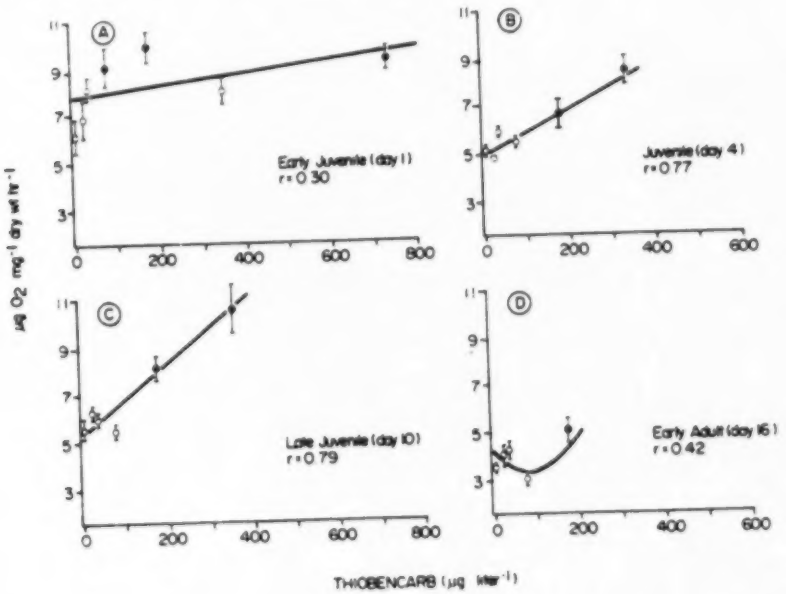

Fig. 2. Respiration rates of various life stages of Mysidopsis bahia as a function of continuous thiobencarb exposure through a complete life cycle. Values represent means ± SE. Darkened circles are significantly different from control at 0.05 probability level.

To best illustrate the influence of thiobencarb on respiration of M. bahia throughout its life cycle, the entire data set for respiration was regressed on the quadratic function of day in life cycle and thiobencarb concentration as described in Equation 2 ($r = 0.73$). The coefficients of this function (Table 1) were used to develop the response surface shown in Figure 3. The response curves depict the dominant influence of the linear interaction between day in life cycle and thiobencarb concentration and the significant main effect of each factor acting individually on mysid respiration. Even though weight-specific respiration rates of juvenile mysids decreased as they developed, the stimulation of mysid respiration by thiobencarb exposure became exaggerated with prolonged exposure.

Table 1. Coefficients for models describing the physiological responses of Mysidopsis bahia to thiobencarb exposure throughout a complete life cycle. Abbreviations of model terms are those described for Equation 2.

Physiological Response	Model Term	Coefficient
Oxygen consumption ($\mu g\ O_2$/mg dry wt/h)	Intercept	7.045042
	CONC	0.006978
	$CONC^2$	-0.000006
	DAY	-0.171730
	DAY^2	-0.004321
	CONC x DAY	0.000737
O:N ratio	Intercept	57.052402
	CONC	-0.084752
	$CONC^2$	0.000053
	DAY	-3.316112
	DAY^2	0.097934
	CONC x DAY	0.005928
Growth (μg dry wt/d)	Intercept	-8.625074
	CONC	-0.139513
	$CONC^2$	0.000330
	DAY	9.666398
	DAY^2	-0.262703
	CONC x DAY	-0.013189
Net growth efficiency (K_2 values)	Intercept	89.174004
	CONC	-0.096446
	$CONC^2$	-0.000162
	DAY	-2.709149
	DAY^2	0.088125
	CONC x DAY	0.006211

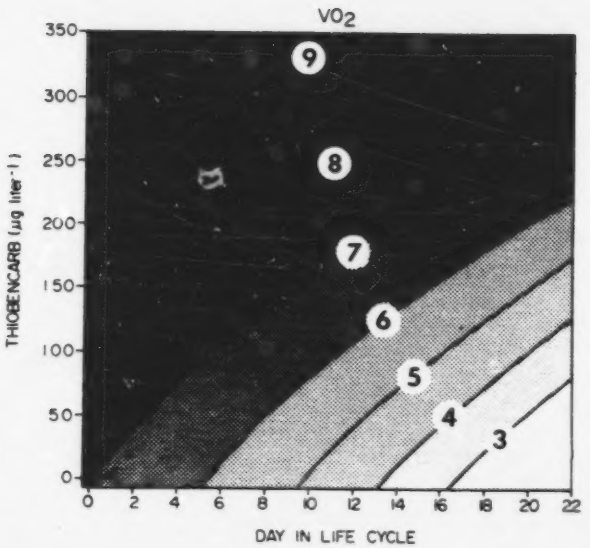

Fig. 3. Response surface of respiration rates (μgO_2/mg dry wt/h) of Mysidopsis bahia as influenced by continuous thiobencarb exposure through a complete life cycle.

Because the two-way analysis of variance of the ammonia excretion data showed a highly significant ($p = 0.0002$) interaction between life stage of exposed mysids and exposure concentration, ammonia excretion rates of individual life stages were regressed against thiobencarb concentration (Fig. 4). After 24 h of exposure, ammonia excretion rates of juveniles were not significantly elevated by thiobencarb exposure. Quadratic regressions best fit the excretion data of older juveniles, for which lower thiobencarb concentrations had no effect and exposure to a concentration of 345 µg/L significantly ($p<0.05$) increased ammonia excretion. As juveniles matured, ammonia excretion rates were reduced with exposure to lower thiobencarb concentrations, a significant reduction occurring at 75 µg/L.

Even though a two-way analysis of variance of log-transformed O:N ratios for thiobencarb-exposed mysids demonstrated a highly significant ($p = 0.0001$) influence by

thiobencarb and life stage individually and a less significant ($p = 0.08$) influence by the interaction of life stage and thiobencarb, linearity of O:N ratios to thiobencarb concentration was poor (Table 2). Nevertheless, O:N ratios of juveniles exposed for at least 4 d to 345 µg thiobencarb/L were significantly ($p<0.05$) reduced. O:N ratios of adults were significantly lower than those of juveniles. However, these adult O:N ratios were significantly elevated in thiobencarb concentrations above 35 µg/L.

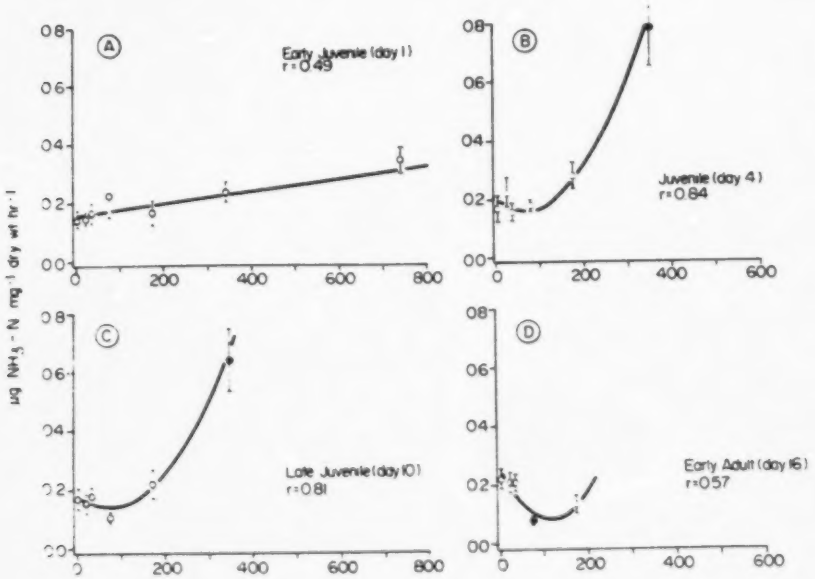

Fig. 4. Ammonia excretion rates of various life stages of Mysidopsis bahia as a function of continuous thiobencarb exposure through a complete life cycle. Values represent means ± SE. Darkened circles are significantly different from control at 0.05 probability level.

These relationships between O:N ratios of different mysid life stages and thiobencarb exposure are best illustrated by the response surface of O:N ratios (Fig. 5) produced from coefficients of the quadratic function of day in life cycle and thiobencarb exposure concentration ($r = 0.43$) (Table 1). This surface depicts the depressed O:N ratios of

exposed mysid juveniles, the reduction of O:N ratios as juveniles mature, and the elevation of adult O:N ratios with thiobencarb exposure.

Table 2. Influence of thiobencarb exposure on O:N ratio of different life stages of Mysidopsis bahia continually exposed through an entire life cycle. Values are the means and standard errors.

Thiobencarb Concentration (µg/L)	O:N Ratio			
	Early Juvenile	Juvenile	Late Juvenile	Early Adult
<0.50 (control)	42 ± 13	35 ± 8	36 ± 10	15 ± 2
22	55 ± 12	30 ± 10	53 ± 15	20 ± 4
35	50 ± 8	41 ± 5	34 ± 5	20 ± 3
76	62 ± 17	31 ± 3	48 ± 7	*36 ± 6
181	72 ± 20	23 ± 3	43 ± 11	*48 ± 20
345	36 ± 5	*10 ± 2	*17 ± 3	-
734	30 ± 5	-	-	-

* Significantly different from the control at 0.05 probability level.

Because a two-way analysis of variance showed a highly significant ($p = 0.0001$) interaction between growth of particular life stages and thiobencarb concentration, daily growth rates of mysids were regressed across thiobencarb concentrations for each phase in the life cycle (Fig. 6). For all life stages, a quadratic regression best fit the growth data, indicating the curvilinear nature of the response to thiobencarb concentration. Exposure concentrations from 22 to 345 µg thiobencarb/L significantly ($p<0.05$) reduced growth rates of juvenile mysids. Retardation of growth rates by thiobencarb exposure was most severe during the most rapidly growing period in the life cycle (day 4 to day 10). As juveniles approached maturity, growth rates of unexposed mysids were reduced. However, growth of juveniles, that was previously retarded in low sublethal concentrations of thiobencarb, was significantly enhanced at these same concentrations as they matured into adults.

Net growth efficiency values (K_2 values) of mysids were significantly ($p = 0.0001$) influenced by an interaction between life stage and thiobencarb exposure concentration. Regressions of K_2 values during different life stages demonstrate these relationships (Fig. 7). Growth efficiency of mysids during the first 4 d of their life cycle was significantly ($p<0.05$) reduced by thiobencarb exposure (≤22 µg/L) in a highly significant linear fashion ($r = 0.98$). The sensitivity of this response was less as juveniles continued to grow. As mysids matured, growth efficiency was significantly increased in sublethal thiobencarb concentrations that had previously reduced efficiency of juveniles.

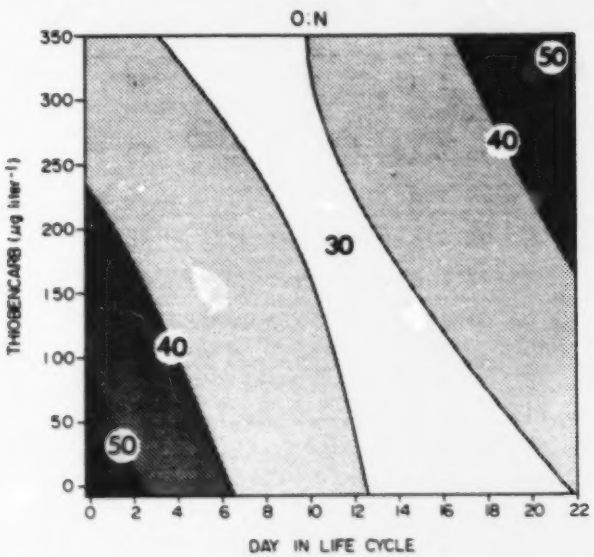

Fig. 5. Response surface of O:N ratios of Mysidopsis bahia as influenced by continuous thiobencarb exposure through a complete life cycle.

The changing relationships between net growth efficiency of mysids and thiobencarb exposure during a complete life cycle of M. bahia are best depicted by the response surface (Fig. 8) produced from coefficients of the regression of all K_2 values onto the quadratic function of day in life cycle and thiobencarb concentration ($r = 0.86$) (Table 1). This surface illustrates the direct inhibition of growth efficiency during the early juvenile period, the reduced growth efficiency of mysids as they mature, and the diminished influence of thiobencarb on growth efficiency as mysids mature.

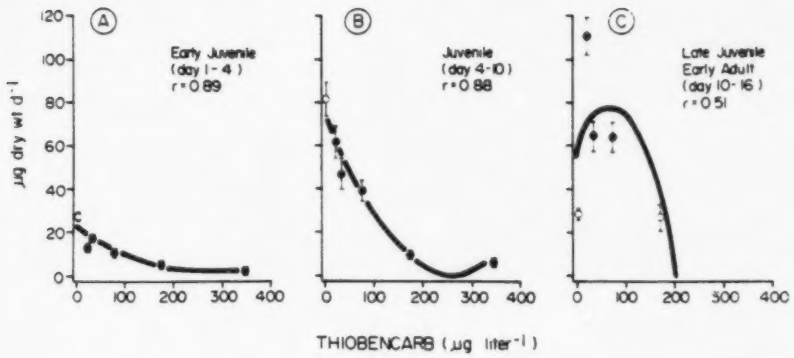

Fig. 6. Growth rates of various life stages of Mysidopsis bahia as a function of continuous thiobencarb exposure through a complete life cycle. Values represent means ± SE. Darkened circles are significantly different from control at 0.05 probability level.

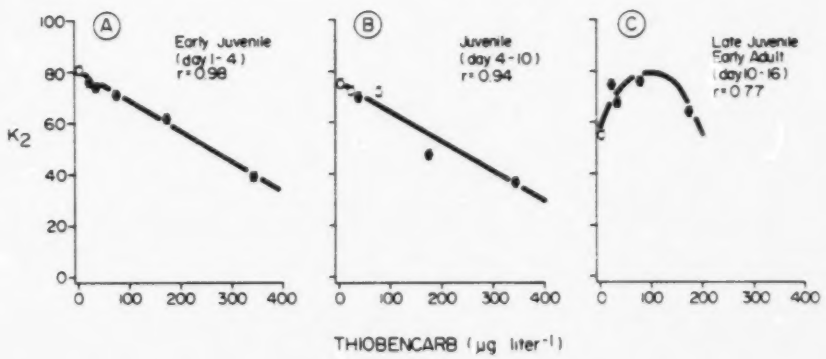

Fig. 7. Net growth efficiencies (K_2 values) of various life stages of Mysidopsis bahia as a function of continuous thiobencarb exposure through a complete life cycle. Values represent means ± SE. Darkened circles are significantly different from control at 0.05 probability level.

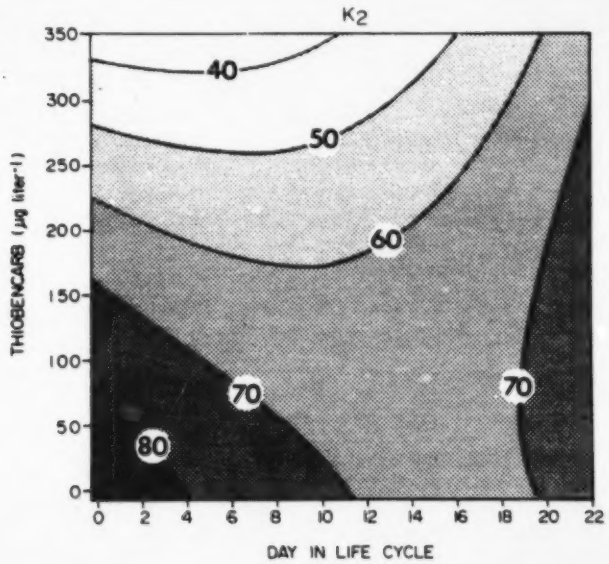

Fig. 8. Response surface of net growth efficiencies (K_2 values) of Mysidopsis bahia as influence by continuous thiobencarb exposure through a complete life cycle.

DISCUSSION

Chronic exposure of Mysidopsis bahia through an entire life cycle to the organic pesticide, thiobencarb, produced lower survival rates and inhibited the reproductive success of the population at sublethal concentrations. Production of young by individual females was inversely and linearly related to exposure concentration. The inherently high variability in this response, however, resulted in it being no more sensitive statistically to thiobencarb exposure than lethality. Nevertheless, total production of young by the population, representing a summation of the effects of these reduced survival and individual fecundity rates on the reproductive success of the population, was inhibited by concentrations an order of magnitude below those concentrations which were acutely lethal for this species (96-h LC50 = 330 µg thiobencarb/L) (Schimmel et al., 1983). These findings are in agreement with previous studies that have demonstrated inhibited reproduction to be the most sensitive sublethal, population response of this species to chronic pesticide exposure (McKenney, 1982 and references therein).

Metabolic alterations in juvenile M. bahia exposed to thiobencarb preceded population-level responses. Respiration rates of juvenile mysids were significantly elevated by acute exposure to the same concentrations of thiobencarb that ultimately led to significant reductions in population survival rates with chronic exposure through an entire life cycle. Thiobencarb stimulation of mysid respiration may be a result of functional disruptions of cytochrome oxidases, as has been described with other thiocarbamates (Ashton & Crafts, 1981), or a result of oxidative metabolism by the microsomal mixed-function oxidases that degrade adsorbed thiobencarb (Hubbell & Casida, 1977; Casida, 1978; Lee, 1981). Regardless of the mechanism, higher respiration rates of juvenile mysids might be useful in the laboratory and, given adequate baseline information, in the field to predict contamination that would produce lowered survival rates of the population with chronic exposure. Since similar correlations were reported between juvenile respiration and population survival for M. bahia exposed to an organochlorine pesticide (endrin) (McKenney, 1982), the utility of this response may apply to many classes of toxic organics. Furthermore, results of a study by Vargo (1981) suggest that metabolic alterations in marine zooplankton may have implications for long-term ecological disruptions at even higher levels of biological organization. Altered patterns of respiration and excretion of individual zooplankters (of separate species) immediately after exposure to low concentrations of oil in an enclosed microcosm led to structural changes in the community after 6-wk exposure.

O:N ratios for the various life stages of M. bahia indicated a shift in the energy metabolism toward greater catabolism of protein as juveniles matured. Increased catabolism of proteins may reflect metabolic shifts of lipids from use as an energy source to mobilization and transfer to developing gonads as a prerequisite for successful gametogenesis. O:N ratios obtained for M. bahia adults were similar to those reported for the adults of several other marine mysids (Chin, 1974; Gaudy et al., 1980). In addition, similar shifts in energy metabolism previously have been reported for maturing mysids (McKenney, 1982) and for reproductively active copepods (Conover & Corner, 1968; Bamstedt & Matthews, 1975). Mobilization, storage, and utilization of lipids during reproductive preparations have been reported for other marine zooplankton (Lawrence, 1976; Gatten et al., 1979).

Modifications in the energy metabolism of individual mysids exposed to thiobencarb were related to reproductive inhibitions, with long-term implications on the ecological fitness of the mysid population. Ammonia excretion rates, although stimulated concurrently with metabolic rates at chronically lethal thiobencarb concentrations, were neither as sensitive to thiobencarb exposure nor as meaningful in biological interpretation as when combined with oxygen consumption rates into an O:N ratio. An apparent changing pattern of substrate utilization during the maturation of M. bahia was altered by exposure to high sublethal concentrations of thiobencarb. Higher O:N ratios during the maturation of these thiobencarb-exposed mysids suggest a greater reliance on lipid material as a substrate for oxidative metabolism, which would result in less lipid material being available for reproductive preparations. Similar relationships existed between energy metabolism and reduced reproductive success of M. bahia exposed to endrin (McKenney, 1982).

It should be noted that both ammonia excretion rates and O:N ratios of mysids were more variable than respiration rates, and neither parameter was as sensitive to thiobencarb exposure as respiration rates. Assurance of the nutritional state of each organism by feeding immediately prior to metabolic measurements might stabilize the observed variation in these measurements and strengthen their dose-response patterns. Similar coordination of feeding and metabolic measurements could result in higher respiration rates reflective of maximum early post-absorptive metabolism (J. Capuzzo, pers. comm.). Regardless of these potential sources of error in absolute values, the relative influence of thiobencarb exposure on the bioenergetics of M. bahia remains the same.

Retarded growth rates of juvenile mysids exposed to thiobencarb were accompanied by decreasing net growth efficiencies, suggesting that the increased metabolic demands of exposed mysids, reflected in the elevated respiration rates, allowed for less assimilated energy to be available for somatic production. Similarly, elevated respiration rates of juvenile M. bahia exposed to endrin resulted in retarded growth rates (McKenney, 1982).

Other marine zooplankton have shown disruptions in bioenergetic processes following sublethal exposure to toxic organics. Acute exposure to oil reduced ingestion rates and elevated respiration rates of adult planktonic shrimp (Lee et al., 1978), but caused significant reductions in the

respiration and excretion rates of larval lobsters (Capuzzo & Lancaster, 1982). Juvenile mysids (M. almyra), exposed to a drilling mud-sea water mixture for a week, displayed higher rates of ingestion and respiration, accompanied by reduced growth rates and lowered growth efficiencies (Carr et al., 1980). Crab larvae reared in oil-treated sea water exhibited reduced rates of ingestion and growth, while respiration rates were increased (Johns & Pechenik, 1980). None of these studies, however, examined bioenergetic processes of the various life stages exposed throughout an entire life cycle; therefore, precluding the establishment of dose-response relationships for these physiological responses and their comparison with long-term alterations in population responses of ecological significance (i.e., survival, growth, and reproduction).

Dose-response relationships for different physiological responses of pesticide-exposed mysids varied with life stage. The more linear relationships between respiration and thiobencarb concentration observed in juvenile mysids resulted in lower net growth efficiencies and retarded growth rates of juveniles. The absence of an inhibitory effect by thiobencarb on growth efficiencies of maturing juveniles, combined with reproductive inhibitions at these same concentrations, imply that as juveniles mature the physiological effects of thiobencarb exposure influence reproductive processes more than growth. Furthermore, thiobencarb exposure affected substrate utilization patterns of maturing juveniles more than those of earlier juveniles. These changing patterns were correlated with lower reproduction rates, suggesting that higher lipid catabolism of mysids maturing in sublethal thiobencarb concentrations may be the mechanism responsible for inhibited reproductive capacities of adult mysids. Additional research involving detailed analysis of biochemical composition could provide supportive information in this area.

Alterations in environmental quality which ultimately modify the normal structural and functional properties of an ecosystem are a product of population and community changes that result from initial damage at the organismic and suborganismic level (NAS, 1971; Sprague, 1971; Waldichuk, 1979; Kinne, 1980; Sastry & Miller, 1981). Environmental modifications of physiological activity of individuals may reflect distortions in ecological mechanisms, such as secondary production. Lower secondary production in a crustacean population, as indicated in this study by retarded mysid growth rates and inhibited reproduction rates, would alter

the energy-flow patterns between connected trophic levels in the ecosystem. Although thiobencarb is directly toxic to fish in low mg/L concentrations (Ochiai & Kubota, 1978; Johnson & Finley, 1980; Metelev & Brichko, 1980; Schimmel et al., 1983), the reduced secondary production of mysid populations with exposure to low µg/L concentrations of thiobencarb could indirectly reduce fish populations that depend upon mysids as a food source (Darnell, 1958; Odum, 1971; Carr & Adams, 1973; Adams, 1976; Livingston, 1980) and disrupt the delicate balance of marine food webs.

From the responses of M. bahia to chronic thiobencarb exposure through an entire life cycle, it may be concluded that metabolic dysfunctions in individual mysids preceded population responses important in determining community tropic patterns. Lower secondary production rates of the mysid populations, manifested by retarded growth rates in juveniles and inhibited reproductive performance in adults, were preceded by modifications in the energy metabolism of individual mysids. Integration of several individual functional responses into physiological and bioenergetic indices proved to be more sensitive than individual physiological measurements for assessing environmentally-induced alterations in biological fitness. Short-term measurements of altered metabolic patterns in contaminated zooplankton, therefore, may offer the potential for monitoring and predicting ecological disruptions in the marine ecosystem at higher levels of biological organization (i.e., the population or community level) (Fig. 9).

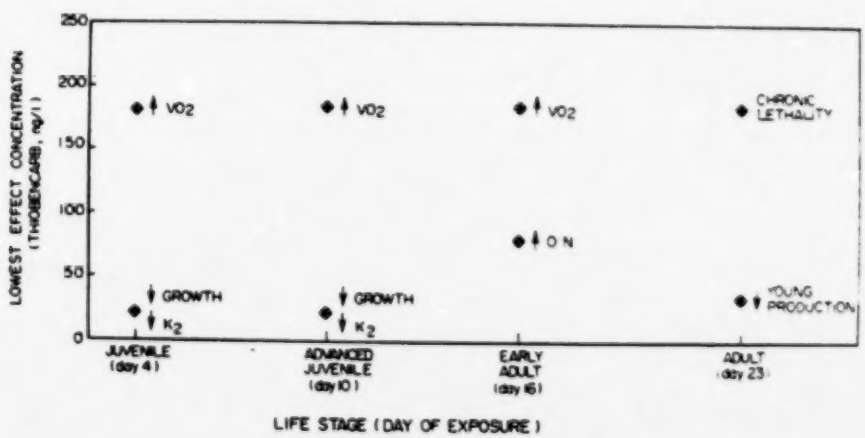

Fig. 9. Summary of the effects of thiobencarb on Mysidopsis bahia exposed through an entire life cycle.

ACKNOWLEDGMENTS

The valuable assistance of Ed Matthews, Debra Lawrence, Neil Santarella, and Meta Leckband during portions of this study is greatly acknowledged. Special thanks are extended to Jim Moore and Johnnie Knight for their quantitative analyses of thiobencarb.

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